MBL and MoBL

A recent 'online early' section of the *British Journal of Haematology* included a paper entitled 'Diagnostic criteria for monoclonal B-cell lymphocytosis' (Marti *et al*, 2005), which is a consensus document on the approach to individuals with low levels of monoclonal B cells.

Low level monoclonal B cells can be detected in a substantial fraction of healthy individuals and the biology and the management of this condition is of considerable significance. The authors suggest the use of the abbreviation 'MBL' for this condition.

Since 1991 the term MBL has been used as an abbreviation for mannose-binding lectin. A search in Pubmed using the terms 'MBL mannose' gave 349 publications on this molecule. MBL is the initial component of what now is called the lectin pathway of complement activation. The molecular properties, polymorphisms and defects of the MBL have been described and many clinical studies have linked MBL to susceptibility to infection and to other inflammatory conditions.

Hence, the use of MBL for the low levels of monoclonal B cells is bound to cause some confusion in the literature. I therefore recommend to amend this abbreviation and suggest that MoBL should be used to denote monoclonal B-cell lymphocytosis.

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Reference

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MBL and MoBL – Response to Ziegler-Heitbrock

We would like to thank Professor Ziegler-Heitbrock for bringing to our attention the prior use of MBL as an abbreviation for 'mannose-binding lectin' and its role in the lectin pathway of complement activation. During the consensus process, monoclonal B-cell lymphocytosis (MBL) was selected from a very large range of alternatives. It is extremely difficult to find a relevant nomenclature with an acronym that has not been used previously. The acronym that you suggest – MoBL – has already been applied to several different medical terms (Drolet & Lau, 1992; Grattan *et al*, 1992; Yoshida *et al*, 1999; Inui *et al*, 2003) and may equally cause confusion. Furthermore, the acronym MBL has been used at international workshops and conferences for more than a year now, and several centres already use this term to refer to the presence of monoclonal B cells in otherwise healthy individuals. We would therefore prefer that the original term MBL is maintained.

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Inherited *FANCD1/BRCA2* exon 7 splice mutations associated with acute myeloid leukaemia in Fanconi anaemia D1 are not found in sporadic childhood leukaemia

Acute myeloid leukaemia (AML) is an uncommon haematological malignancy in childhood, accounting for approximately 10–15% of all childhood leukaemias. However, children affected by Fanconi anaemia (FA) have a substantially increased risk of developing AML (Alter, 2003). FA is a rare inherited chromosomal fragility syndrome, in which the multiprotein pathway that appears to play a major role in the recognition and repair of DNA damage (D'Andrea & Grompe, 2003) is disrupted. At least 11 genes can be mutated in FA including *FANCA-C*, *FANCD1/BRCA2*, *FANCD2*, *FANCE-G*, *FANCL* and the so far unidentified genes for subtypes FA-I & J (Levitus *et al*, 2004). Although all FA patients have an increased risk of AML, patients of the rare FA-D1 group develop AML earlier in life (Howlett *et al*, 2002). FA-D1 patients have biallelic mutations in the breast cancer susceptibility gene, *BRCA2*, and mutation analysis has revealed a striking correlation between mutation type and early onset AML. Of 23 FA-D1 patients reported in the literature, nine developed AML within the first 3 years of life (Table I). All AML patients had an IVS7 splice site mutation in one or both alleles.

To address the question of the role of IVS7 splice site mutations in susceptibility to sporadic (i.e. non-FA) associated AML, we scanned constitutional DNA from 90 children with this leukaemia. Genomic DNA was extracted from remission blood samples that were collected between 1992 and 1996 in various centres in the UK. A diagnosis of AML was confirmed by haematological review panels as part of the treatment protocols. One-third of the patients were <5 years old. For comparison we included 50 children with T-cell and 50 with B-cell precursor ALL.

Table I. *FANCD1/BRCA2* mutations associated malignancy reported in FA-D1 patients.

	Genotype	
Patient code and malignancy	Allele 1	Allele 2
IFAR 129/1 AML	IVS7 + 2T>G	IVS7 + 2T>G
IFAR 632/1 AML	IVS7 + 1G>A	5910C>G
IFAR 632/2 AML		
IFAR 800/1 AML	IVS7 + 2T>G	5146del4
IFAR 800/2 Wilm's occult		
SB 1690CB AML	IVS7+2>G	3827delGT
IFA 3R 57/1 AML	8106 G>C	2041insA
AP 37P AML	8415G>T	8732C>A
EUFA 579 AML	7235G>A	5837TC>AG
Kin 2-1 Wilm's, AML	4876G>T	7757T>C
Kin 2-2 T-ALL		
IFAR 900/1 T-ALL	2816insA	1342C>A
Kindred 1/1 posterior fossa	6174delT	9435T>A
Kindred 1/2 astrocytoma		
Kindred 2 medulloblastoma	6174delT	886delGT
Kindred 3 medulloblastoma	5301insA	7690T>C
Kindred 4 medulloblastoma	4150G>T	9424C>T
IFAR 772/1 medulloblastoma	886delGT	8447T>A
IFAR 772/2 Wilm's, medulloblastoma		
HSC62 No cancer at age 30 years	IVS19-1G>A	IVS19-1G>A
EUFA 432 brain tumour	7691insAT	9900insA
*Wilms2-RB Wilm's, glioblastoma	886delGT	5873C>A
*Wilms2-CB Wilm's, medulloblastoma, B-ALL		

Meyer *et al*, 2005, *Reid *et al*, 2005